11:45

PROTEIN KINASE C ACTIVITY AND ITS SUBCELLULAR DISTRIBUTION IN THE ATRIA AND VENTRICLES OF RATS WITH CARDIAC HYPERTROPHY.

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An increased Ca^{2+}-mediated inotropic activity is observed in the early stages of experimental cardiac hypertrophy (CH). Ca^{2+} plays a key role in the inositol trisphosphate-diacylglycerol-protein kinase C transduction system (PK-C). To assess the potential relationship between PK-C and CH, we studied the PK-C activity, determined by histone phosphorylation with P^{32}-ATP in partially purified heart extracts (cytosolic and particulate fractions) of aorto-caval shunt-induced heart hypertrophy (3 weeks; n=5) and that of normal rats (n=5). Total and cytosolic enzymatic activities were 67 and 91% higher, respectively, in the hypertrophied hearts, p< .05 vs controls. Particulate PK-C activity decreased 25% (p< .05). In each cardiac chamber and particularly in the left ventricle, the cytosolic activity was 3 times higher in hypertrophied hearts.
In summary, our results shown that PK-C activity is increased in CH. These findings could be related to the inotropic changes observed in experimental CH.

LACK OF BENEFIT OF EARLY ROUTINE INVASIVE STRATEGY IN REDUCING REINFARCTION RATE IN HIGH-RISK PATIENTS AFTER THROMBOLYSIS (Results of a randomized trial: SIAM-I).

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324 pts with acute myocardial infarction (AMI) were treated with intravenous streptokinase (< 4 hrs after onset of symptoms, 1.500.000 U/1hr) in 13 acute care hospitals and were centrally randomized by phone call to the university hospital in two groups during fibrinolysis:
GROUP A: (invasive strategy) CA with PTCA/CABS 14 to 48 hours after start of treatment and predischarge control CA.
GROUP B: no CA within the first 21 days, unless there is evidence for ischemia and predischarge CA.
Serial creatine kinase (CK) determinations at least every 4 hrs were available in 255 pts, 47/255 pts (18%) suffered from re-AMI (overall reinfarction rate: 56/324 pts (17%)). The CK peak was found to be the best predictor of reinfarction. In pts with CK maximum (CKmax) < 400 U/l the incidence of reinfarction was 23/70 (33%) compared to those with CKmax > 400 U/l 24/185 (13%; p < 0.01).
Reinfarction in gr A and gr B:

Pts with:	gr A	gr B	p
CKmax > 400 U/l	7/ 94(7%)	17/ 91(19%)	0.05
CKmax < 400 U/l	8/ 26(31%)	15/ 44(34%)	n.s.

CONCLUSION: Patients with low CK maximum are at high risk for reinfarction after fibrinolytic treated AMI. A routinely performed invasive strategy does not reduce reinfarction rate in this high-risk subgroup of patients.

Wednesday, March 6, 1991
Poster Displayed: 9:00AM–12:00NOON
Author Present: 9:00AM–10:00AM
Hall F, West Concourse
Thrombolytic Therapy II

THE ROLE OF COLLATERAL CIRCULATION IN THE OCCURRENCE OF POSTINFARCTION ISCHEMIA FOLLOWING THROMBOLYSIS

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Although it is well known that collateral circulation (CC) often develops following acute myocardial infarction, little information exists about its relationship with the occurrence of postinfarction (PI) ischemia in pts submitted to thrombolysis (TL). The present study was undertaken to examine the correlation between the evidence of CC and PI ischemia in 86 pts with myocardial infarction who received i.v. TL. All pts underwent coronary angiography within one month from admission; 26 pts had early PI angina, whereas 29 showed a positive exercise stress testing (ST shift >0.1 mV). At angiography an adequate CC was present in 22/33 pts who had an occluded infarct related artery (IRA), and only in 5/53 pts with patent IRA (67% vs 9%; p<0.001). PI angina was more frequent in pts with CC (15/27) than in those without (11/59): 56% vs 19%, p<0.05. A positive exercise response was present in 9/11 pts with occluded IRA and CC, but only in 14/38 pts with patent IRA and no CC (82% vs 37%; p<0.05). Thus: (1) CC is uncommon in pts with evidence of successful TL; (2) development of CC is associated with the occurrence of PI angina; also, its presence correlates with the induction of ischemia during exercise in pts with IRA occlusion; (3) myocardial salvage of perinfarctual zones by CC is suggested to be a cause of recurrent PI ischemia.

THE PROPORTION OF MYOCARDIUM AT RISK SALVAGED BY REPERFUSION THERAPY IS INDEPENDENT OF INFARCT LOCATION

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To investigate the influence of infarct location on myocardial salvage, 38 pts with first myocardial infarction (MI) were injected with Tc-99m-Isonitrile (RP30A) prior to acute reperfusion therapy given within 6 hours of the onset of chest pain (primary coronary angioplasty (PTCA): 20 pts, thrombolysis: 18 pts) and again at hospital discharge (6-14 days later). Tomographic imaging (SPECT) was performed within 8 hours of injection. Perfusion defect size was quantitated as a percent of the left ventricle with absent perfusion using previously reported methods. Acute and discharge defect size indicated the myocardium at risk (RISK) and infarct size (INF), respectively. Myocardial salvage (SALV) was defined as the change in defect size from the acute study to discharge. Salvage index was defined as SALV/RISK, representing the proportion of RISK salvaged. Radionuclide ejection fraction (EF) was obtained at discharge.

		Anterior n=21	Inferior n=17	P
Acute defect	RISK	56 ± 10%	21 ± 11%	<.0001
Discharge defect	INF	31 ± 21%	12 ± 9%	.001
Change in defect	SALV	25 ± 17%	9 ± 8%	<.002
Salvage Index		0.46 ± 0.34	0.48 ± 0.35	NS
Final EF		0.39 ± 0.14	0.56 ± 0.09	<.0001

Conclusions: Pts with anterior MI have greater RISK, greater INF, greater myocardial salvage, and lower EF. However, the proportion of RISK which is salvaged, estimated by salvage index, is nearly identical for anterior MI and inferior MI and approaches 1/2 of the myocardium at risk.